JACC Vol 2, No. 3
September 1983:488–96

# Immediate Diagnosis of Acute Myocardial Contusion by Two-Dimensional Echocardiography: Studies in a Canine Model of Blunt Chest Trauma

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**The purpose of this study was to assess the usefulness of two-dimensional echocardiography in the diagnosis of myocardial contusion resulting from blunt chest trauma. In 25 anesthetized dogs, blunt chest trauma was delivered to the right or left chest using a captive bolt pistol. Eighteen dogs survived. Two-dimensional echocardiographic recordings and myocardial perfusion determinations (microspheres) were performed before and 15 and 90 minutes after trauma. Pathologic examination confirmed and localized the extent of myocardial contusion. When trauma was delivered to the left side of the chest, two-dimensional echocardiographic and pathologic abnormalities were primarily in the anterolateral wall of the left ventricle; right chest trauma produced septal and right ventricular wall contusion.**

**From short-axis two-dimensional echocardiographic recordings, the end-diastolic wall thickness and systolic function of regions that corresponded to areas of damage in anatomic sections were analyzed and compared with those of remote normal-appearing regions. The contused region showed increased end-diastolic wall thickness and impaired regional systolic function. Further, there was a visible increase in the echo brightness in the contused region. Regional perfusion was normal and thus the functional impairment of the contused myocardium was not due to ischemia. Other abnormalities observed on the two-dimensional echocardiograms included pericardial effusion and discrete intramyocardial sonolucent zones due to hematomas.**

**Contused myocardium can be identified on a two-dimensional echocardiogram by 1) increased end-diastolic wall thickness, 2) increased echo brightness, and 3) impaired regional systolic function. Two-dimensional echocardiography can also demonstrate other complications of cardiac trauma such as intramyocardial hematoma and hemopericardium.**

Myocardial contusion is a frequent and sometimes lethal result of nonpenetrating chest injuries (1–5). The diagnosis of myocardial contusion is often difficult because of coexisting injuries and because of the nonspecificity of diagnostic tests, such as electrocardiography, serum enzyme determination, chest radiography and radionuclide scanning (6–10). The role of two-dimensional echocardiography in cardiac trauma is not established; because echocardiography can examine the entire heart in multiple tomographic planes and ultrasonic equipment can easily be moved to and used in an emergency setting, two-dimensional echocardiography is potentially valuable in the evaluation of cardiac trauma. The purpose of our study was to assess the usefulness of two-dimensional echocardiography in the diagnosis of myocardial contusion resulting from blunt chest trauma.

## Methods

We used two-dimensional echocardiography in a canine model of myocardial contusion. Twenty-five mongrel dogs, weighing 14 to 28 kg, were studied.

**Animal preparation.** The dogs were anesthetized with intravenous sodium pentothal (500 mg) and chloralose (100 mg/kg body weight), intubated and ventilated using a Har-

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vard volume respirator. Polyurethane catheters were placed into the right and left ventricles, the abdominal aorta, the right brachial artery and the femoral artery and vein for pressure measurements and fluid and drug administration. A lead II electrocardiogram was monitored throughout the procedure.

*We produced blunt chest trauma and myocardial injury* by firing a captive bolt gun against the intact chest wall of the anesthetized dog. The method has been described in detail by Anderson and Doty (11). A Super-Cash pistol (Accels and Shelvoke, Ltd.), originally designed for the abattoir industry, was modified by attaching a 2 inch (5 cm) semielliptical steel disc to the end of the force transmitting piston. When a 0.22 caliber blank cartridge was discharged, the pistol was propelled forward, with a nominal velocity of 480 ft (144 m)/s. Injury results from the transfer of kinetic energy through the chest wall to the underlying myocardium. In 17 dogs, the device was fired with the piston positioned firmly against the left chest wall slightly above the point of maximal cardiac impulse. This resulted in contusions confined primarily to the left ventricle. In eight other dogs, in order to induce right ventricular trauma, the blow was delivered to the right chest wall.

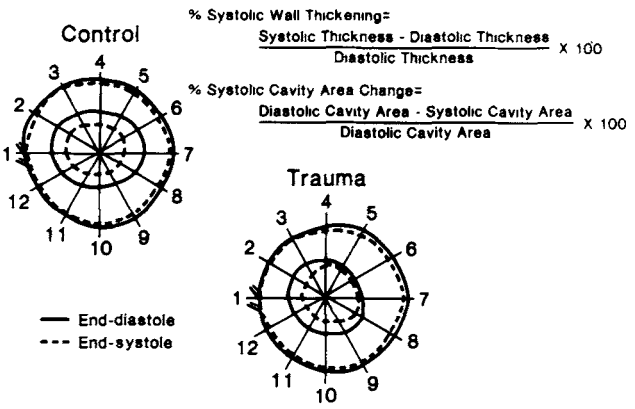



**Figure 1.** Method used for analysis of regional left ventricular contraction in short-axis two-dimensional echocardiographic recordings. The posterior junction between the right ventricular free wall endocardium and the right side of the interventricular septum was chosen as a landmark, and a line was drawn to the farthest point on the lateral left ventricular wall epicardium (**line 1—7** in the illustration). This line was designated as a cavity diameter, and bisected to identify the cavity center. Twelve equidistant radii were drawn to separate the image into 12 cavity and wall segments. Radii 1 to 5 correspond approximately to the interventricular septum, 5 to 9 to the anterolateral left ventricular wall and 9 to 1 to the posterior left ventricular wall. Percent thickening of the wall along each radius and percent area change of each cavity segment during systole were calculated. After trauma, an increase in end-diastolic wall thickness and a decrease in systolic wall thickening and cavity area change occurred in the contused region (radii 4 to 9 in this example).

**Echocardiographic studies.** Two-dimensional echocardiograms were performed using a Toshiba SSH-10A sonolayergraph, utilizing a 2.4 MHz phased-array transducer. Using the technique of Wyatt et al. (12), the dogs were placed on a specially constructed examination table on their right side with the right chest wall positioned over a window in the table. The hand-held echo transducer was directed upward against the right chest wall and tomographic images were recorded in the long-axis and in four short-axis planes, using internal landmarks for orientation: the mitral valve, the chordae tendineae, the papillary muscles and the left ventricular apex (that is, below the papillary muscles). Images were recorded on videotape for later real-time slow-motion and stop-frame analysis. Our time references for stop-frame analysis were end-diastole, defined as the peak of the electrocardiographic R wave, and end-systole, defined as the minimal apparent cross-sectional cavity area. The end-diastolic and end-systolic epicardial and endocardial boundaries from short-axis images were traced from stop-frames using transparent plastic sheets placed over the video monitor. For consistency, we always traced the inner borders of the endocardium and epicardium. The tracings of the epicardial and endocardial boundaries in the short-axis images were then digitized using a Talos digitizing tablet, interfaced to a PDP-11/34 computer and Ramtek image display system.

*The method of echocardiographic analysis* is standard in our laboratory and has been described in detail previously (13). Briefly, a landmark in each image was the posterior junction of the endocardium of the right ventricular free wall and the right side of the ventricular septum (Fig. 1). We defined the ventricular diameter as the longest line from this landmark (in diastole) to the left ventricular anterolateral wall epicardium. This line divided the ventricle approximately in half. The midpoint of this diameter was used as the center of the short-axis image. From this center, 12 radii at 30° intervals were generated, dividing the short-axis image into 12 ventricular cavity and ventricular wall segments. Numbered clockwise, the radii 1 to 5 correspond approximately to the ventricular septum, 5 to 9 to the anterolateral and 9 to 1 to the posterior left ventricular wall. In each short-axis tomographic section, the end-diastolic and end-systolic length of each radius was measured.

*Percent systolic thickening of the wall along each radius was calculated by the equation:*

$$\frac{\text{End-systolic wall thickness} - \text{End-diastolic wall thickness}}{\text{End-diastolic wall thickness}} \times 100.$$

*Percent segmental cavity area change in each segment was calculated using the equation:*

$$\frac{\text{End-diastolic cavity area} - \text{End-systolic cavity area}}{\text{End-diastolic cavity area}} \times 100$$

**Myocardial perfusion measurement.** Regional myocardial perfusion was measured with radioactive-labeled microspheres using standard techniques (14). We use 15$\mu$ microspheres labeled with $^{141}$cerium, $^{85}$strontium, $^{46}$scandium, $^{95}$niobium or $^{113}$tin. For each perfusion measurement, the vial containing the microspheres was mechanically agitated for at least 3 minutes to disperse the spheres. Twenty $\mu$ Ci microspheres were injected over a 5 second period into the left ventricular catheter; the catheter then was flushed with 10 ml of saline solution. Beginning 1 minute before injection and continuing for 3 minutes thereafter, blood for reference flow determinations was withdrawn from the right brachial and femoral arteries simultaneously at 2.06 ml/min. No hemodynamic alterations occurred as a result of sphere injection. At the end of the experiment, the dogs were rapidly killed with potassium chloride. Two myocardial samples in each dog, about 1 g each, were obtained from the visibly contused area of the left ventricle. Additional samples were taken from the normal-appearing myocardium opposite the visibly contused area. Using techniques previously described (14), the perfusion of each myocardial segment was determined.

**Pathologic examination.** At the conclusion of each experiment, each dog was killed and an immediate autopsy performed. Hemopericardium or pericardial tears were noted. The heart was then removed and examined for external injuries. After fixation in 10% formaldehyde for a day, the heart was cut into four transverse tomographic sections to correspond to the short-axis echo images. The contused areas were mapped by gross inspection (Fig. 2). Other lesions, if present, were noted.

**Experimental protocol.** After the dogs were anesthetized and catheters were inserted, control hemodynamic and echocardiographic recordings were performed. The dogs were then separated into two groups: Group I (17 dogs) received blunt chest trauma to the left side of the chest; Group II (8 dogs) received the trauma to the right side of the chest. In nine of the dogs in Group I, microspheres were injected for control perfusion determination. Intravenous lidocaine, 50 mg, was given to all dogs as a bolus injection about 2 minutes before the trauma. The animals all developed transient ventricular tachyarrhythmias and brief periods of hypotension immediately after trauma. They were resuscitated with electrical defibrillation, using 100 joules delivered energy for initial shocks and higher energies if necessary for ventricular fibrillation. Additional lidocaine was given for ventricular tachycardia and intravenous saline and epinephrine solution (2 ml, 1:10,000) for hypotension. If the dogs survived the initial effects of the trauma, their hemodynamic status usually became stable in about 10 minutes.

*Hemodynamic, echocardiographic and perfusion assessments were repeated 15 minutes after the trauma.* In addition, four dogs in Group I and four others in Group II were further observed until 90 minutes after trauma, and echographic and hemodynamic recordings and perfusion measurements were repeated. Regional perfusion was not measured in Group II. After the experiment was complete, pathologic examination and perfusion determinations were performed as noted earlier.

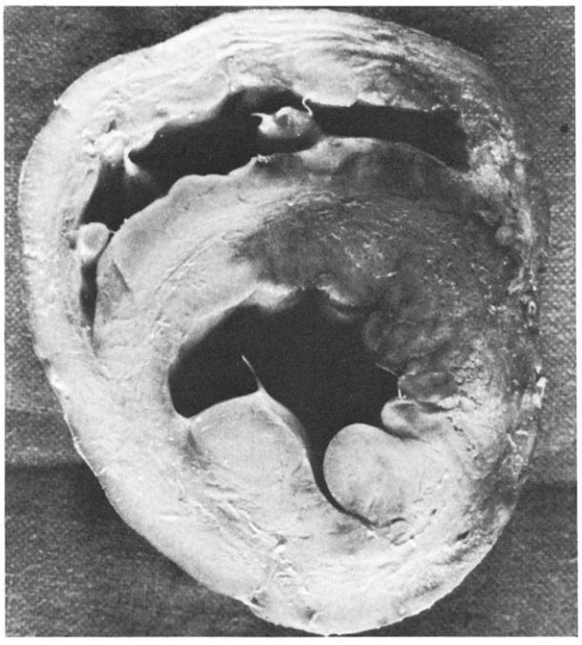


**Figure 2.** Myocardial contusion (left chest trauma). Transverse cut section of the left and right ventricles at the level of the papillary muscles. The contused area (**dark area**) involves the anterolateral left ventricular wall, anterior half of the interventricular septum and adjoining right ventricle.

**Data analysis.** Groups I and II were analyzed separately. Hemodynamic measurements and regional perfusion changes of normal and contused myocardium at 15 and 90 minutes after trauma were compared with the control values using analysis of variance. The echocardiographic data analysis was done as follows:

1) *Qualitative echocardiographic changes of myocardial contusion.* We evaluated the ability of observers to diagnose myocardial contusion solely on the basis of grossly visible echocardiographic appearance. Two observers were shown one known control and one known post-trauma recording in a training session. Then they were shown, in a blinded fashion, a series of recordings that included (in random order) one control and one post-trauma image from each animal. The two observers were asked to diagnose the recording as being normal or showing myocardial contusion. The simultaneous electrocardiographic recordings were masked to deemphasize premature ventricular beats which were often present in the post-trauma recordings (although observers could still suspect them from the echographic recordings themselves). Using this information, the sensi-

tivity (true positive ÷ true positive + false negative results), specificity (true negative ÷ true negative + false positive results) and predictive accuracy (true positive ÷ true positive + false positive results) of the echocardiographic technique in the diagnosis of myocardial contusion were determined for each blinded observer.

2) *Quantitative echocardiographic changes of myocardial contusion.* We determined the quantitative effects of myocardial contusion on regional myocardial function, as demonstrated by two-dimensional echocardiography. End-diastolic wall thickness, percent systolic wall thickening and percent systolic cavity area change were analyzed in echocardiographic short-axis images that corresponded to the anatomic sections exhibiting pathologic evidence of myocardial contusion. Initially, the extent of contusion was mapped by gross inspection of the pathologic slices. Then, corresponding segments were identified in the echocardiographic recordings using landmarks such as the papillary muscles and anterior and posterior junction of the interventricular septum with the right ventricular wall. The pathologic and echocardiographic segments opposite the contused region, which showed no evidence of damage, were identified as the normal region. The echocardiographic measurements obtained at 15 and 90 minutes in the contused region and in the remote normal-appearing region were compared separately with control measurements. All comparisons were made using analysis of variance.

A probability (p) value less than 0.05 was considered significant. Data are expressed as mean ± 1 standard deviation.

## Results

Of the 25 dogs studied, 7 died immediately after the trauma: 4 of intractable ventricular fibrillation, 2 of right atrial tear with hemopericardium and cardiac tamponade and 1 of severe pump failure. Four other dogs had ventricular fibrillation but were successfully resuscitated. All dogs that survived the trauma had brief periods of ventricular tachycardia but usually their rhythm became stable with restoration of sinus rhythm by 10 minutes after trauma. ST segment elevation was noted in seven dogs, but was transient, lasting 2 to 5 minutes after trauma. Overall, 12 dogs in Group I and 6 dogs in Group II survived to have follow-up recordings.

**Hemodynamic changes (Table 1).** Although all dogs had severe ventricular arrhythmias in the first 1 to 2 minutes after trauma, the heart rhythm stabilized with lidocaine and, when necessary, defibrillation. The heart rate was then similar to the control heart rate, at both 15 and 90 minutes after trauma. Left and right ventricular end-diastolic pressure increased after trauma while left ventricular systolic pressure and aortic pressures decreased. Despite these statistically significant changes, the pressures remained within physiologic ranges; none of the surviving animals were severely hypotensive or in shock when the post-trauma recordings were obtained. There was no difference in hemodynamic response between Groups I and II.

**Pathologic findings.** All the dogs that survived the immediate catastrophic effects of trauma had evidence of myocardial contusion characterized by localized edema and ecchymosis and, in four dogs, intramyocardial hematoma. The contusion was often transmural with a greater extent at the epicardial surface (Fig. 2). Contusion generally occurred in the anteroseptal and anterolateral regions of the left ventricle and the adjacent right ventricle in Group I, which received left chest trauma. The dogs in Group II, which received right chest trauma, had contusions involving the right ventricular free wall and the interventricular septum. In addition to the two dogs that died of right atrial tear and cardiac tamponade, three other dogs had hemopericardium without evidence of tamponade. No evidence of injuries to the valves or coronary arteries was found on gross pathologic examination in any dog.

### *Echocardiographic Findings*

**Qualitative echocardiographic changes of myocardial contusion.** There were abnormal echocardiographic findings in all dogs in which two-dimensional echocardiographic study could be performed after trauma. In addition to the two dogs that died of cardiac tamponade, three surviving dogs showed two-dimensional echocardiographic evidence of fluid (blood) in the pericardium.

On two-dimensional echocardiograms, the contused myocardial area appeared swollen; systolic wall thickening and motion were decreased after trauma. There was a localized increase in the brightness of the intramyocardial echoes (Fig. 3 and 4). In addition, an echolucent area surrounded by an area of increased echo intensity was seen in each of four dogs that had an intramural hematoma (Fig. 4). These abnormal echographic findings were seen in the areas corresponding to the areas of contusion noted on pathologic examination: the anterolateral wall and the anterior portion of the interventricular septum in Group I dogs, primarily the interventricular septum in Group II dogs. Right ventricular wall thickness was noticeably increased after trauma in one Group II dog; the right ventricle could not be visualized satisfactorily in the other dogs using this experimental model. Valvular structures appeared normal in all the dogs.

The sensitivity, specificity and predictive accuracy of two blinded observers in diagnosing myocardial contusion by two-dimensional echocardiography were 97, 86, and 88%, respectively (Table 2).

**Quantitative echocardiographic changes of myocardial contusion (Table 3).** *End-diastolic wall thickness.* The end-diastolic wall thickness was increased in the contused region at 15 and 90 minutes after trauma in both Group

**Table 1.** Hemodynamic Effects of Blunt Chest Trauma in 18 Dogs (mean ± standard deviation)

| | Control | 15 Minutes After Trauma | 90 Minutes After Trauma |
|---|---|---|---|
| Heart rate (beats/min) | 135 ± 18 | 143 ± 15 | 136 ± 14 |
| Right ventricular pressure (mm Hg) | | | |
| Systolic | 24 ± 5 | 26 ± 6 | 28 ± 6 |
| End-diastolic | 1 ± 1 | 4 ± 3* | 4 ± 4* |
| Left ventricular pressure (mm Hg) | | | |
| Systolic | 146 ± 25 | 106 ± 21* | 105 ± 15* |
| End-diastolic | 5 ± 3 | 10 ± 4* | 7 ± 1† |
| Aortic pressure (mm Hg) | | | |
| Systolic | 146 ± 24 | 100 ± 25* | 105 ± 15* |
| Diastolic | 101 ± 21 | 64 ± 20* | 69 ± 18* |

*$p < 0.05$ versus control; †$p < 0.05$ versus 15 minutes after trauma.

I (Fig. 5) and Group II. The noncontused normal myocardium did not show any change.

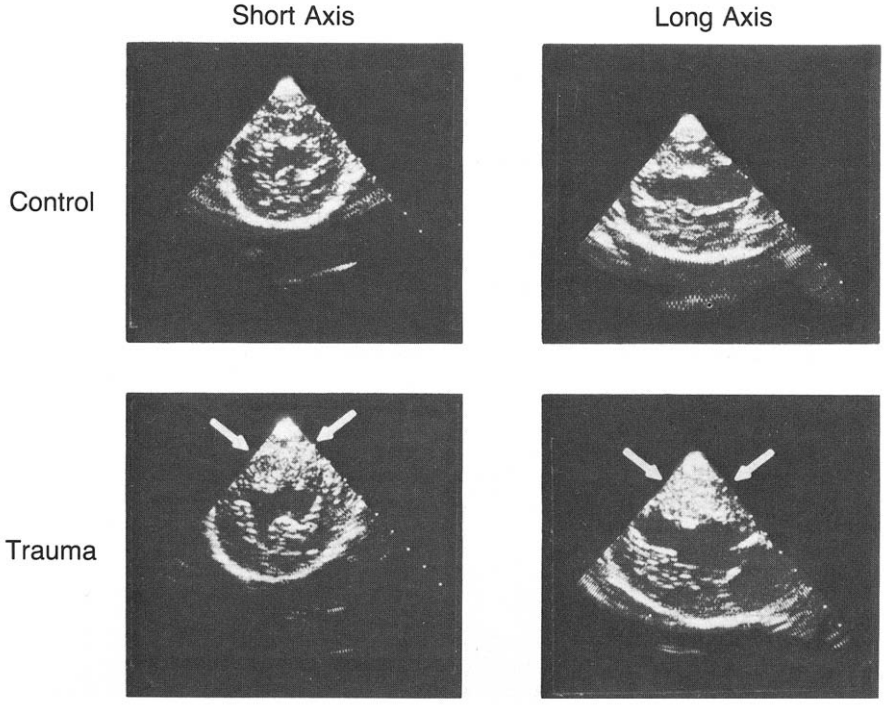
*Regional left ventricular systolic function.* Two indexes of regional left ventricular systolic function, percent systolic wall thickening and percent systolic cavity area change, were analyzed. After trauma, there was a decrease in both these indexes in Group I (Fig. 6 and 7) and Group II (Table 3).

*Regional myocardial perfusion (Table 3).* Control transmural perfusion was normal. The perfusion tended to be higher in the contused region and the normal region 15 minutes after trauma, but the difference did not achieve statistical significance. The blood flow returned to control levels, both in normal and contused segments by 90 minutes after trauma.

## Discussion

**Echocardiographic features of myocardial contusions.** This study demonstrates that two-dimensional echocardiography can identify and localize the structural and functional abnormalities of myocardial contusion resulting from blunt chest trauma. The distinctive echocardiographic features of regional myocardial contusion are: 1) increased end-diastolic wall thickness, 2) impaired contraction, indicated by decreased percent systolic wall thickening and

**Figure 3.** Short- and long-axis two-dimensional echocardiographic recordings from a dog before **(top panels)** and 90 minutes after **(bottom panels)** right chest trauma. Increased brightness and increased wall thickness are noted in the septum **(arrows).**



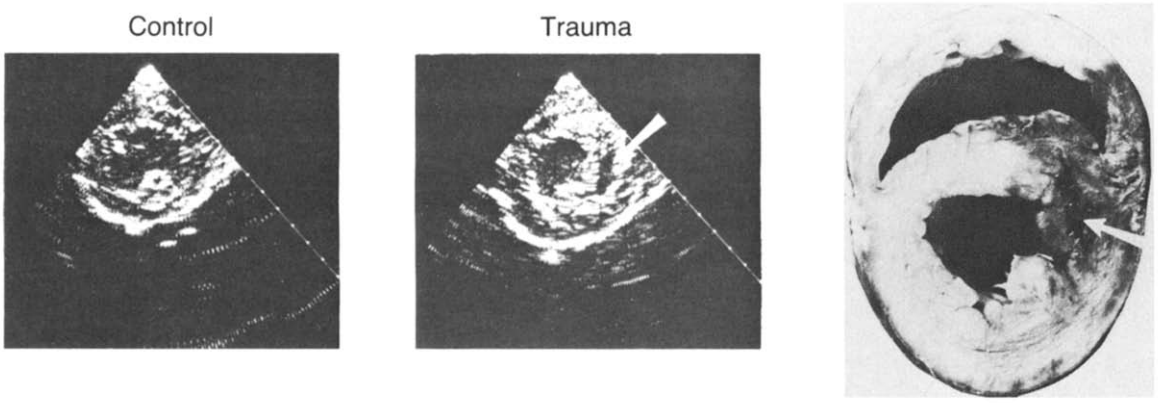



**Figure 4.** Short-axis two-dimensional echocardiographic recordings at the level of the papillary muscles from a dog before and 15 minutes after left chest trauma. Wall thickness is increased in the contused area. Within the bright echo-dense area, a discrete, linear, sonolucent zone is seen, representing an intramural hematoma **(arrow).** A transverse cut section of the pathologic specimen in the same dog shows the contusion **(dark area)** and the hematoma **(arrow).**

decreased percent systolic cavity area change, 3) increased brightness of the ultrasonic reflections from the contused areas, and 4) discrete localized areas of echolucency when intramural hematomas were present. In addition, associated complications, such as hemopericardium, are detectable. Our experiments have also shown that regional myocardial blood flow remains normal in the contused region and thus hypoperfusion or ischemia is not responsible for impairment of regional function.

*Common causes of nonpenetrating injury to the heart in civilian life* include automobile accidents, especially deceleration injuries where the victim is thrown forward against the steering wheel or dashboard, direct blows to the chest by any kind of blunt object or missile (such as a clenched fist or sporting equipment) kicks of large animals (such as horses and cattle) and falls (1–5). The lesions produced in our model were comparable with those observed by pathologic examination in human subjects after severe closed chest trauma (3,15,16).

**Table 2.** Sensitivity, Specificity and Predictive Accuracy of Two-Dimensional Echocardiography in the Diagnosis of Myocardial Contusion

| | Sensitivity (%) | Specificity (%) | Predictive Accuracy (%) |
|---|---|---|---|
| Observer A | 94 | 94 | 94 |
| Observer B | 100 | 78 | 82 |
| Total | 97 | 86 | 88 |

n = 36 echocardiographic recordings (18 control and 18 post-trauma).

*Cardiac injury is the most common unsuspected visceral injury responsible for death in fatally injured accident victims (3).* In penetrating chest injuries, surgical exploration is usually undertaken and, thus, internal injuries are accurately detected. In contrast, blunt nonpenetrating chest trauma may present with scant evidence of cardiac damage and a myocardial contusion may be easily missed or overlooked as attention is directed to associated rib fractures, flail chest and lung injuries. Even if contusion is suspected, lack of reliable diagnostic techniques makes it difficult to establish its presence (10). Clearly, there is a need for a noninvasive approach that can readily detect myocardial contusions and other cardiac damage. Two-dimensional echocardiography is readily available for emergency use and provides repeated real-time examination, but its role has not been established in this setting. The present study indicates how two-dimensional echocardiography could be used to make the diagnosis of myocardial contusion.

**Correlation of echocardiographic and pathologic myocardial changes.** The echocardiographic abnormalities noted in our experimental study parallel the pathologic changes resulting from cardiac contusion. Edema in the injured area explains the increased end-diastolic wall thickness seen on echocardiography. Contractile function is impaired by any form of myocardial injury. Increased brightness is probably due to altered tissue architecture secondary to cell damage and hemorrhage, with multiple fibrin-blood-tissue interfaces. Abnormal tissue seen in other conditions, such as hypertrophied interventricular septum in idiopathic hypertrophic cardiomyopathy and infiltrative myocardium in amyloidosis, also shows increased "speckle" and brightness on two-dimensional echocardiographic images (17,18). Similarly, altered echo amplitude has been found after experimental myocardial infarction (19). In our experiments, when there was a circumscribed cavitational area within the myocardium filled only with blood, a localized echolucent zone consistent with a hematoma was seen. Such echolucent patterns have been previously described in patients with intra-

**Table 3.** Effects of Blunt Chest Trauma on Echocardiographic and Perfusion Measurements in 18 Dogs (mean values ± standard deviation)

| | Control | 15 Minutes After Trauma | 90 Minutes After Trauma |
|---|---|---|---|
| Group I (n = 12 dogs) | | | |
| Echocardiographic features | | | |
| End-diastolic wall thickness (mm) | | | |
| Normal region | 8 ± 1 | 9 ± 2 | 9 ± 2 |
| Contused region | 8 ± 1 | 15 ± 2*† | 15 ± 2*† |
| % Systolic wall thickening | | | |
| Normal region | 52 ± 14 | 52 ± 24 | 58 ± 22 |
| Contused region | 56 ± 14 | 3 ± 10*† | 7 ± 14*† |
| % Systolic cavity area change | | | |
| Normal region | 45 ± 14 | 47 ± 13 | 56 ± 19 |
| Contused region | 52 ± 14 | 16 ± 37*† | 35 ± 30 |
| Perfusion (ml/100 g per min) | | | |
| Normal region | 111 ± 34 | 131 ± 70 | 100 ± 44 |
| Contused region | 90 ± 38 | 145 ± 68 | 103 ± 53 |
| Group II (n = 6 dogs) | | | |
| Echocardiographic features | | | |
| End-diastolic wall thickness | | | |
| Normal region | 7 ± 1 | 7 ± 1 | 8 ± 1 |
| Contused region | 7 ± 1 | 12 ± 3*† | 15 ± 3*† |
| % Systolic wall thickening | | | |
| Normal region | 75 ± 40 | 77 ± 22 | 59 ± 17 |
| Contused region | 71 ± 16 | 8 ± 9*† | 9 ± 6*† |
| % Systolic cavity area change | | | |
| Normal region | 54 ± 20 | 48 ± 15 | 47 ± 13 |
| Contused region | 59 ± 14 | 9 ± 45*† | 24 ± 31* |

*$p < 0.05$ compared with control; †$p < 0.05$ compared with normal region.

myocardial hemorrhage during acute myocardial infarction (20).

**Potential limitations of study.** All of our dogs required resuscitation, frequently including defibrillation, after trauma. We questioned whether some of the two-dimensional echocardiographic changes we saw could have been related to these resuscitative maneuvers or to brief periods of ischemia during resuscitation, rather than directly to the trauma. We previously performed many echocardiographic studies of experimental ischemia and infarction without blunt chest trauma, and we defibrillated and resuscitated numerous dogs during these studies (13,14,19). Systolic contraction abnormalities due to ischemia were universal in dogs subjected to ischemia, defibrillation and resuscitation, but we never

**Figure 5.** Effect of trauma on end-diastolic left ventricular wall thickness. The contused segments show a localized increase in wall thickness immediately after the trauma. The remote segments remain unchanged in thickness.

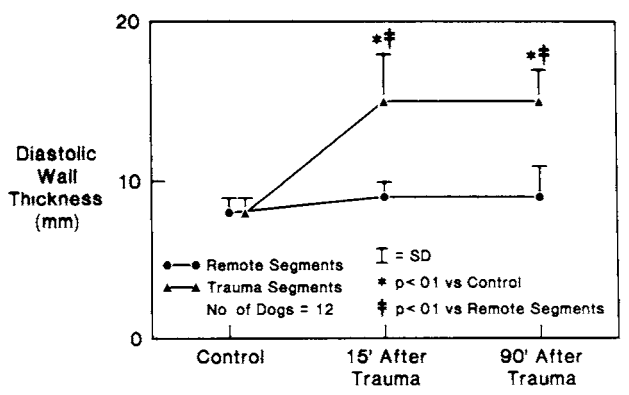
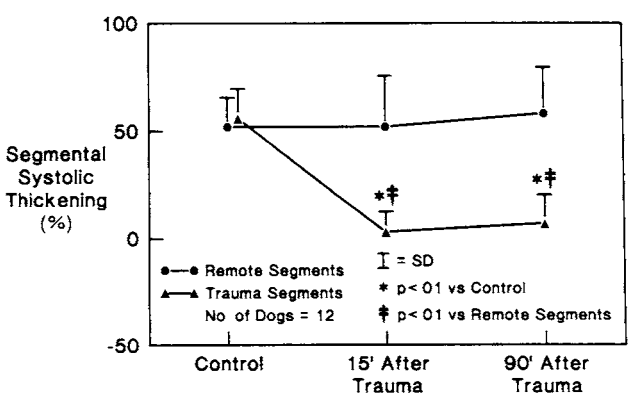



**Figure 6.** Effect of trauma on percent systolic left ventricular wall thickening. After trauma, the contused segments show a significant impairment of this variable. The remote segments retain normal function.



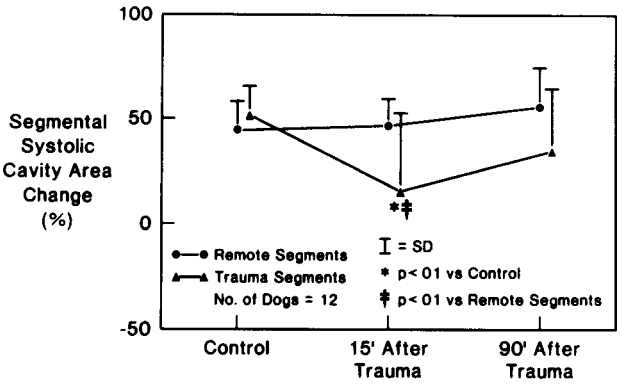



**Figure 7.** Effect of trauma on percent systolic cavity area change. Immediately after trauma, the contused segments show a significant reduction in this variable. The remote segments retain normal function.

saw increased brightness and increased end-diastolic thickness in those animals without trauma. Moreover, sensitive techniques such as sonomicrometers have not demonstrated contractile changes in dogs undergoing closed chest defibrillation alone, without trauma (21). Thus, it seems very unlikely that the two-dimensional echocardiographic changes we saw after trauma were related primarily to the resuscitative efforts or to brief periods of ischemia that may have occurred during resuscitation.

In our closed chest dog model we were usually not able to obtain images of right atrium and right ventricle of adequate quality for quantitative analysis. The right ventricular anterior wall was noted to be thickened after trauma in one dog and it is likely that the right ventricular myocardium, if it had been adequately visualized in other dogs, would have manifested findings similar to those seen in the left ventricle. In a report of seven patients studied several days after chest injuries due to automobile accidents, Miller et al. (22) emphasized changes of right ventricular myocardial size and function.

*The cardiac injury produced was relatively severe.* We wondered if two-dimensional echocardiography could also detect less severe injuries. In several pilot studies (11), we attempted to produce milder injuries by using a smaller cartridge in the pistol, resulting in a lower piston velocity. However, we found that using such lower energy trauma produced minimal echocardiographic and pathologic changes. Thus, our model and the echographic findings we report may only be applicable to more severe forms of cardiac trauma; whether mild trauma is detectable by two-dimensional echocardiography remains to be demonstrated.

**Regional myocardial perfusion and role of ischemia.** Myocardial perfusion tended to show an increase early after trauma, although this change did not achieve statistical significance. Our results are consistent with the experimental observations made by previous workers who also noted increased, not decreased, flow in the injured zones (23,24). Thus, ischemia does not appear to occur and is not likely to be the mechanism for the diminished regional left ventricular function in contusion (2,8,23,24). Why myocardial perfusion tends to increase in both contused and normal regions, in the face of a decrease in coronary perfusion pressure, was not established by our study. Complex neural and humoral mechanisms in the regulation of the coronary circulation may be involved. Edema and hemorrhage in the contused area probably make the myocardium tense, less compliant and less contractile, which are probable reasons for the observed changes in regional systolic function after trauma.

We observed no coronary artery injuries in the dogs we studied. Likewise, in human beings, coronary artery injuries, although occasionally reported, are not common in blunt chest trauma (25).

In our perfusion studies, microspheres were injected into the left ventricle, avoiding the thoracotomy that a left atrial cannulation would have required. However, the accuracy of coronary blood flow determinations using microspheres depends on the uniform mixing of microspheres in the bloodstream. Wicker and Tarazi (26) compared left ventricular and left atrial injection sites for coronary blood flow measurements; they found that the mean flow values from both approaches were not significantly different, although the dispersion of the flow measurements was two-fold higher with left ventricular injections. We showed previously that ischemic or infarcted myocardium typically has blood flow values in the range of 15 to 25% of control (14,27,28). Thus, the somewhat greater variability of flow measurements from left ventricular microsphere injections would not be enough to mask the marked drop in perfusion that would have been present with ischemia, if ischemia were the cause of the post-trauma echographic changes. Because we did not see such a drop in myocardial perfusion, ischemia is very unlikely.

**Clinical implications.** Cardiac contusion is a major clinical problem. Our experimental study suggests that two-dimensional echocardiography will be useful in identifying myocardial contusion immediately after trauma. Abnormalities in our study were seen early after trauma and lasted for at least 90 minutes. Because patients with chest injuries are frequently brought to the hospital within 90 minutes, two-dimensional echocardiography may be a valuable diagnostic approach to the detection of myocardial contusion in patients sustaining blunt chest trauma.

We thank Diane Phillips and Linda Mohr for their help in the preparation of the manuscript and Anuradha Vaitheswaran for her assistance in the project

## References

1. Bright EF, Beck CS. Nonpenetrating wounds of the heart: a clinical and experimental study. Am Heart J 1935;10:293–321.
2. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958;18:371–96.
3. Liedtke AJ, DeMuth WE Jr. Nonpenetrating cardiac injuries: a collective review. Am Heart J 1973;86:687–97.
4. Symbas PN. Cardiac trauma Am Heart J 1976;92.387–96.
5. Cheitlin MD. Key references: cardiovascular trauma. Circulation 1982,65:1528–32.
6. Ware RE, Martin LG, Tyras DH, Kourias E, Symbas PN Coronary arterial injection of radioactive albumin microspheres in diagnosis of experimental myocardial contusion. Surg Forum 1972;23:138–42.
7. Martin LG, Larose JH, Sybers RG, Tyras DH, Symbas PN. Myocardial perfusion imaging with $^{99m}$Tc-albumin microspheres. Radiology 1973;107.367–72.
8. Chiu CL, Roelofs JD, Go RT, Doty DB, Rose EF, Christie JH. Coronary angiographic and scintigraphic findings in experimental cardiac contusion. Radiology 1975;116:679–83.
9. Downey J, Chagrasulis R, Fore D, Parmley LF. Accumulation of technetium$^{99m}$ stannous pyrophosphate in contused myocardium. J Nucl Med 1977;18:1171–7.
10. Potkin RT, Werner JA, Trobaugh GB, et al. Evaluation of noninvasive tests of cardiac damage in suspected cardiac contusion. Circulation 1982;66.627–31.
11. Anderson AE, Doty DB. Cardiac trauma: an experimental model of isolated myocardial contusion. J Trauma 1975;237–44
12. Wyatt HL, Heng MK, Meerbaum S, et al. Cross-sectional echocardiography. I. Analysis of mathematic models for quantifying mass of the left ventricle in dogs. Circulation 1979;60:1104–13.
13. Pandian N, Kerber R. Two-dimensional echocardiography in experimental coronary stenosis. I. Sensitivity and specificity in the detection of transient myocardial dyskinesis. Comparison with sonomicrometers Circulation 1982;66:597–602.
14. Marcus ML, Kerber RE, Ehrhardt J, Abboud FM. Three-dimensional geometry of acutely ischemic myocardium. Circulation 1975;52:254–62.
15. Kahn MH, Kahn S. Cardiovascular lesions following injury to the chest. Ann Intern Med 1929;2:1013–23.
16 Moritz AR. Pathology of Trauma. Philadelphia: Lea & Febiger, 1942:143–9.
17. Martin RPM, Rakowski H, French J, Popp RL. Idiopathic hypertrophic subaortic stenosis viewed by wide-angle, phased-array echocardiography. Circulation 1979;59:1206–17.
18 Chiaramida SA, Goldman MA, Zema MJ, Pizzarello RA, Goldberg HM. Real-time cross-sectional echocardiographic diagnosis of infiltrative cardiomyopathy. J Clin Ultrasound 1980;8:58–62.
19. Skorton D, Melton H, Pandian N, et al. Detection of acute myocardial infarction in closed-chest dogs by analysis of regional two-dimensional echocardiographic gray level histograms. Circ Res 1983,52:36–44.
20. Werner JA, Speck SM, Greene HL, Janko CL, Gross BM. Discrete intramural sonolucency: a new echocardiographic finding in acute myocardial infarction (abstr). Am J Cardiol 1981;47:404.
21. Kerber RE, Martins JB, Gascho JA, Marcus ML, Grayzel J. Effects of direct-current countershocks on regional myocardial contractility and perfusion. Circulation 1981;63:323–32.
22. Miller FA, Seward JB, Gersh BJ, Tajik AJ, Mucha P Two-dimensional echocardiographic findings in cardiac trauma. Am J Cardiol 1982;50:1022–7.
23. Utley JR, Doty DB, Collins JC, Spaw EA, Wachtel C, Todd EP Cardiac output, coronary flow, ventricular fibrillation and survival following varying degrees of myocardial contusion. J Surg Res 1976;20:539–43.
24. Doty DB, Anderson AE, Rose EF, Go RT, Chiu CL, Ehrenhaft JL. Cardiac trauma. clinical and experimental correlations of myocardial contusion. Ann Surg 1974;180:452–60.
25. Hawthorne JW, Kantrowitz PA, Dinsmore RE, Sanders CA Traumatic myocardial infarction: report of a case with normal coronary angiogram. Ann Intern Med 1967;66:341–3.
26. Wicker P, Tarazi RC. Importance of injection site for coronary blood flow determinations by microspheres in rats. Am J Physiol 1982;242:H94–7.
27. Kerber RE, Marcus ML, Ehrhardt J, Wilson R, Abboud FM. Correlation between echocardiographically demonstrated segmental dyskinesis and regional myocardial perfusion. Circulation 1975;52:1097–104
28 Pandian NG, Kieso R, Kerber RE. Two-dimensional echocardiography in experimental coronary stenosis. II. Relationship between systolic wall thickening and regional myocardial perfusion in severe coronary stenosis. Circulation 1982;66.603–11